Medical Staff Conference

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Syphilis

These discussions are selected from the weekly staff conferences in the Department of Medicine, University of California, San Francisco. Taken from transcriptions, they are prepared by Drs. David W. Martin, Jr., Assistant Professor of Medicine, and Kenneth A. Woeber, Associate Professor of Medicine, under the direction of Dr. Lloyd H. Smith, Jr., Professor of Medicine and Chairman of the Department of Medicine. Requests for reprints should be sent to the Department of Medicine, University of California, San Francisco, CA 94143.

DR. SMITH:* *The topic for Medical Grand Rounds this morning is one of increasing importance to all physicians. We have asked Dr. Marcus Conant of the Department of Dermatology to discuss the great mimic, syphilis.*

DR. CONANT:† I would like to give you a historical and clinical review of syphilis and in so doing attempt to make two essential points. The first is that we are experiencing a pandemic of syphilis in the world today and that this pandemic is centered primarily in large metropolitan areas such as San Francisco. The incidence of venereal disease, and particularly syphilis, in this community is appalling. This University of California, San Francisco, is one of the largest medical centers in the country and is located in the midst of this epidemic. Yet it would appear that we are doing very little as an institution to assist the community in eradicating syphilis.

The second point that I would like to stress is that syphilis has continually changed in its clinical presentation since it was first described in the early 1500's, and it is continuing to change today. The classical disease that was described by de Villalobos and others back in the early 1500's is no longer seen. What de Villalobos described was an acute febrile disease that suddenly erupted in a community. Large numbers of victims were suddenly struck down with a great pox that spread rapidly over the patient, producing great morbidity and frequently causing death. The syphilis that we see today is far more insidious, frequently is discovered with no history of a chancre, and is often devoid of all but the mildest cutaneous eruption. More and more frequently, secondary syphilis is being misdiagnosed as hepatitis or mononucleosis.

In 1800, Lord Byron wrote a couplet more prophetic than poetic: "The smallpox has gone out of late, Perchance it will be followed by the great." It was not in Byron's time, however, but in ours that his prophecy was borne out. The reported incidence of syphilis in the United States was about 10,000 cases a year in the period 1920 to 1930. In the late 1930's the incidence began to climb, and during the early war years of the 1940's, the reported incidence reached about 35,000 cases a year.[1]

Penicillin was discovered in 1942, and follow-

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ing its introduction into the physician's armamentarium, the incidence of reported cases of infectious syphilis began to fall, decreasing from 35,000 in 1940 to approximately 2,000 in 1960. Then an amazing thing began to happen, for, while new and better antibiotics were being discovered, many of which are very effective in the treatment of infectious syphilis, the incidence of the disease began an exponential rise in early 1960 and continued this increase until, last year, more than 90,000 cases were reported.

There has been a lot of argument about these figures. Some have argued that this is only a reflection of population growth and better reporting. But if one plots a population growth curve against the exponential increase that we have seen with syphilis, the slope of the syphilis curve is certainly more acute than the population growth curve. It has also been argued that reporting today is better than it was forty years ago and that this accounts for the rapid increase that has been seen. Contact work conducted primarily by the U.S. Public Health Service is unquestionably better than it was forty years ago. But if you will recall that before 1940 patients with syphilis were treated with arsphenamine (Salvarsan®) or one of the heavy metal compounds and that they were generally treated in large medical centers over long periods, it becomes obvious that most of those cases were, in fact, reported to the local health authorities. Today, with penicillin readily available, a clinician can make a clinical diagnosis of syphilis, give the patient an injection of penicillin, and yet not report the case to the local authorities who would initiate the proper contact work. In his zeal to treat and care for his patient, the clinician often forgets that, for every case of syphilis he sees, there is another case from which his patient contracted the disease. The public health authorities estimate that only one out of ten to one out of fourteen cases of syphilis in this community are, in fact, reported to them. This, of course, means that the absolute number of cases of syphilis in our community may be ten times higher than that indicated by available figures. The incidence of primary and secondary syphilis in San Francisco rose from 209 reported cases in 1968 to 624 reported cases in 1971. If we compute national incidences by determining the number of cases per 100,000 persons, we arrive at a statistic of 12 cases of syphilis per 100,000 nationally and of 81 per 100,000 in San Francisco.

Numerous reasons have been cited as the primary cause for the increased incidence of syphilis observed in this country in the last ten years. These include promiscuity of the young, sexual experimentation, the birth control pill, increased incidence of homosexuality, changing moral standards, lack of public interest, poor physician training, lack of research funds, social crowding, and (so far as known incidence is concerned) better reporting of contacts. In all probability, no one factor has been responsible for this epidemic. Rather, the problem we now face has come from a concert of many of these factors.

Six Is a Shibboleth in the Stages of Syphilis

For those of you who have not thought much about syphilis for the last few years, let me quickly run through the classical stages of the disease[2] and introduce a concept that I think you will find useful. As to the stages of syphilis, think in terms of sixes. The reason for stressing this will become apparent in a moment. The classical chancre is a nonpainful, indurated, ulcerative lesion frequently located on the penis or cervix, and usually it is associated with regional lymphadenopathy. The lesion usually appears about six weeks after the patient is infected with the disease. The incubation period can be anything from four to ten weeks. The chancre, after it appears, will usually last for a period of from six days to two weeks, and usually after about the sixth day of its appearance the fluorescent treponemal antibody-absorbed (FTA-ABS) test becomes positive.[3] Usually, the Venereal Disease Research Laboratory test (VDRL) becomes positive about the seventh day. This means that the FTA generally becomes positive slightly before the VDRL; and so in the clinical evaluation of early syphilis, one should instruct the laboratory to run both tests, regardless of the reactivity of the VDRL. As you will recall, the chancre begins to disappear at the point that the VDRL becomes positive. This, of course, means that in evaluating an early chancre, the darkfield examination is the only way of making a positive diagnosis. As the chancre is beginning to disappear, the darkfield examination coupled with the VDRL and FTA will establish the diagnosis. A period now elapses between primary and secondary syphilis, and this time can be anything from six weeks to six months. During this period, the titer of the VDRL is rising very rapidly. Classically, a papulosquamous eruption then develops over the patient's entire body, with a papular syphilid of the palms and systemic symptoms including a

low-grade fever, lymphadenopathy, malaise, sore throat, and alopecia. The rash of secondary syphilis usually lasts for about six weeks and then begins to slowly disappear without treatment. Then a long latency period ensues. The latency period may last anywhere from 18 months to 12 years before the patient begins to show signs of tertiary syphilis. Six years is a convenient figure to remember.

History of a Lively Commerce

One way to assist you in making a diagnosis of syphilis is to review the history of the geographic spread of the disease which in itself is a fascinating study and gives an overall view of what has happened to it over the last five hundred years.[4] Syphilis was probably unknown in Europe and Northern Africa until 1493. There are some authorities who still argue that the disease did exist there before that time; but if it did, it existed only as a localized endemic disease and did not cause the epidemic disease that was seen after Columbus returned from the New World. Most medical historians feel that the disease was contracted in the Western Hemisphere from natives and brought back and introduced into southern Spain at the time of Columbus' return in 1493.

Columbus landed south of Seville and travelled to Seville with six Indians and six of his sailors and probably first introduced the disease into that town renowned for oranges, bullfights, and torrid love affairs. Queen Isabella and King Ferdinand were holding court in Barcelona, and so this party of famous adventurers travelled from Seville to Barcelona to present themselves at court. Now you must try to imagine the enthusiasm that greeted Columbus on his return. It was probably greater than the enthusiastic welcome which we gave our astronauts. Columbus had not only sailed away and returned from the edge of the world, but he could bring back natives and artifacts from a new and foreign culture. Unhappily, he also returned with a new disease. (At least in this country, we profited from an admonition which George Santayana has put into graceful words, that those who do not study history are destined to repeat it. When our astronauts returned from the moon, we did place them in an isolation chamber until it could be determined that they were free of any extraterrestrial disease.)

Columbus visited the court of Spain and was well entertained by the powerful and wealthy of the land. The powerful invited Columbus and his troops to their villas and tables, and young ladies bestowed their favors on these adventurous young men. Apparently, the disease spread rapidly and became endemic in southern Spain within a matter of months. A year later, Charles VIII of France was at war with Naples. Naples was held by the Holy Roman Empire and, of course, the Holy Roman Empire at that time included the Hapsburg Empire, Austria, Hungary, Italy, Spain, and Portugal. The city of Naples hired Spanish mercenaries to come and help them fight the King of France. The Spanish armies brought syphilis with them and shared it with the camp followers around Naples, who in turn shared it with the French, and soon the army of France began to show signs of the new disease. The French were successful in their campaign against Naples; but, unfortunately for France, typhus made its European political debut the night after the battle, and the French army was literally decimated before dawn. The French troops had to withdraw to France, taking with them few spoils of war and their newly-acquired disease. This was the period in history when France was expeditionary throughout Europe, and for the next fifty years, the French army spread the disease over the face of the land.

A Reverse Nationalism of Names

Each country named the disease for some other country. In Italy, it was known as "the French disease," in France as "the Italian disease," in England as "the Spanish disease." The first written mention of the disease was by the Diet of Worms in 1495. The Diet issued an edict saying that the disease (which it called *bösen Blattern,* "the evil pox") was infectious and that the patient should be isolated. The edict cautioned that no one should engage in sexual contact with the patient until after the disease had disappeared. This was, of course, before it was realized that the patients were still infectious during the early latent period.

Mercury was first used in 1497 and, of course, heavy metals were the treatment of choice from that time until the early 1940's.

Francisco Lopez de Villalobos wrote the first medical treatise on the disease in 1498. As I have mentioned, he described a disease quite different from that seen today. He said that the first symptoms were usually severe rheumatic pains, fol-

lowed by high fever with prostration and then a rapidly progressing eruption with draining sores over the entire body. The descriptions are more reminiscent of the pustular forms of syphilis than of the papulosquamous forms that are more commonly seen today. The disease was apparently quite catastrophic and spread rapidly through an entire town.

Hieronymus Fracastorius, in 1530, gave the disease the name that we still use. Fracastorius was a very famous Italian physician who practiced and taught in Verona. This was a period in history when the Reformation was challenging the Catholic Church and the Council of Trent was formed to launch the counter-reformation. Fracastorius was chosen by the Pope as the physician to the Council of Trent. An example of the classical Renaissance man, Fracastorius was not only physician and scientist but artist and writer as well, and he used the Renaissance approach to describe the evil pox. He took the famous story of a young shepherd boy who angered the gods and was then struck down by them in vengeance. In telling of the punishment visited on the shepherd, Fracastorius had the boy struck down by a loathsome disease which, described in poetic form, had all of the then recognized manifestations of syphilis. Of course, the name of the young shepherd was Syphilus.

Parenthetically it may be noted that an old wives' tale which has circulated for centuries has it that syphilis, or at least some venereal disease, is contracted from having sexual intercourse with sheep. Man, of course, is the only animal that can contract this disease, and there is no basis in fact for this misconception except that Fracastorius chose a shepherd boy as the hero of this story.

Forty-two years after Columbus returned to Spain, Ruy Diaz de Isla wrote his memoirs. The year was 1539, but de Isla recalled that he had been practicing in southern Spain at the time of Columbus' return and that he had seen some of the sailors from the ship. He recalled that the sailors did suffer from a pox that then rapidly spread throughout the country, and he called the disease "the serpentine disease," not because of its morphological appearance, but because, he said, it was the most loathsome disease he had ever seen. He also said that in his practice he had seen some 20,000 cases of syphilis, which means that he must have been seeing two or three cases a day.

The Four Plus Nonplus of Dr. Hunter

John Hunter was the first to try to debunk the idea that syphilis and gonorrhea had the same cause. In his classical experiment, Hunter took pus from a patient who had gonorrhea and inoculated himself with this material. Unfortunately, he picked the wrong patient, because the patient that he chose as his donor also had syphilis, and Hunter acquired both gonorrhea and syphilis. He died from the latter disease some twenty years later, and his experiment confused the issue for another 30 years. The Scottish physician Dr. Benjamin Bell finally did further experiments and showed that gonorrhea and syphilis were two completely distinct diseases. Dr. Bell did his work on medical students, so it would appear that going to medical school in those days was more perilous than it is today. At least our students do not acquire the disease in the classroom.

From Hunter's study the relevant point to today's clinicians is not to forget that the patient you see with gonorrhea may well have acquired syphilis at the same time, and in evaluating and treating the patient's gonorrhea one must also evaluate and, if necessary, treat for syphilis.

Charcot in 1874 described the gastric crisis of secondary syphilis and the destructive joint changes of tertiary lues. In 1890, Hutchinson described the classic triad of congenital syphilis—notched incisors, eighth nerve deafness, and interstitial keratitis. Two years later, Gene Fournier first proposed the concept of latency. Until then, it had been accepted that when patients recovered from the eruption of secondary syphilis, they had recovered from the disease. It was not known that the disease became latent for a period of six years or so only to resurface later as gummas, neurological lesions, and aortic aneurysms. Fournier's concept of latency was correct. Today, we divide the latent period into two intervals. Early latent syphilis is anytime within two years after the patient recovers from the secondary lesions of the disease. Late latent syphilis is anything more than two years after the secondary eruption.

Carl Landsteiner is a name that you all know from his work on isolating blood group antigens. It is now frequently forgotten that Landsteiner, in 1906, was the first to introduce the darkfield microscope for the detection of treponemes in syphilitic lesions. The same year, August von Wassermann, working with Albert Neisser for whom the gonococcus was named, introduced the

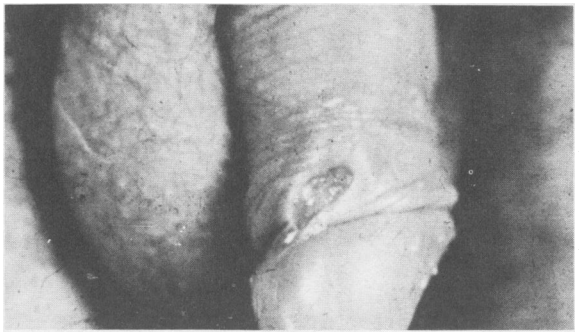

Figure 1.—Chancre

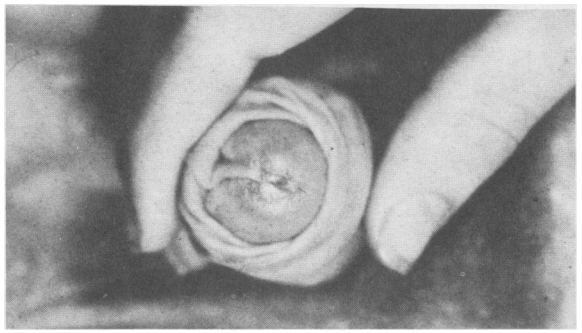

Figure 2.—Chancre of meatus

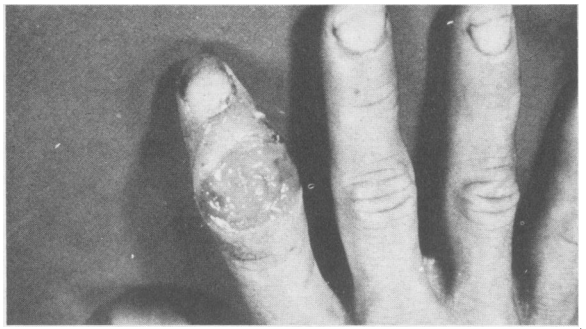

Figure 3.—Chancre of finger

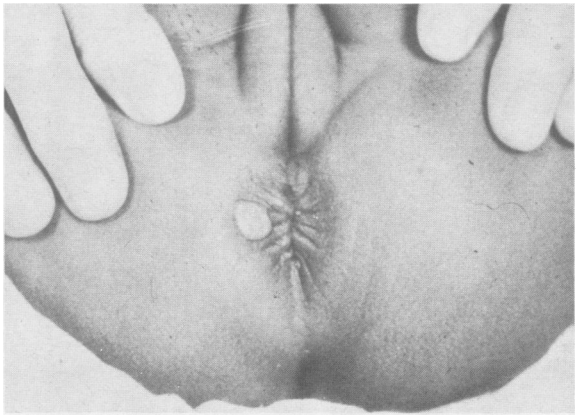

Figure 4.—Anal chancre in child

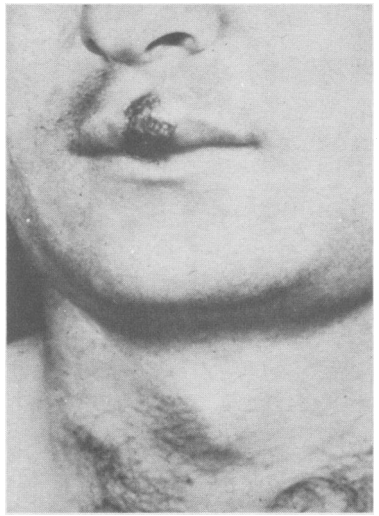

Figure 5.—Chancre of lip simulating herpes simplex

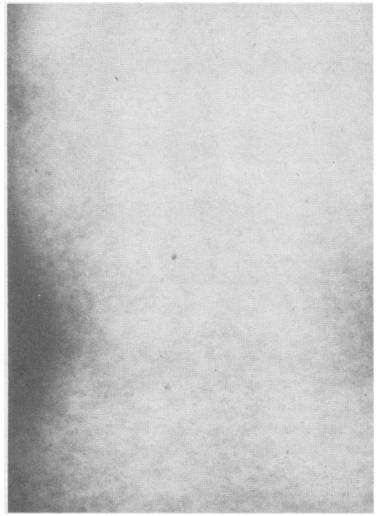

Figure 6.—Macular syphilid

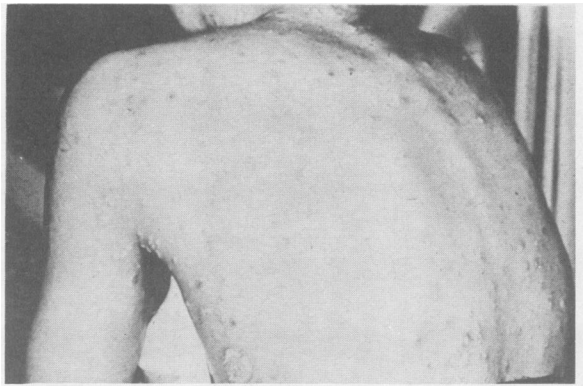

Figure 7.—Papulosquamous lesion of secondary syphilis

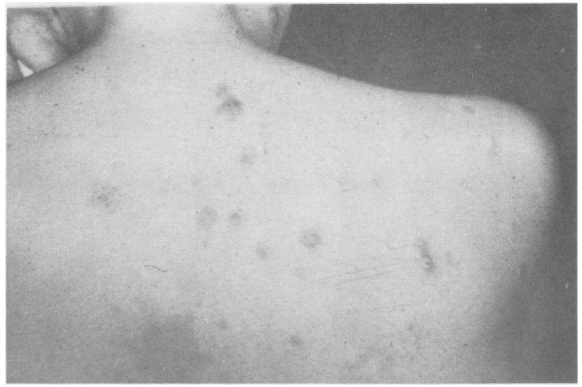

Figure 8.—Papulonodular lesion of secondary syphilis

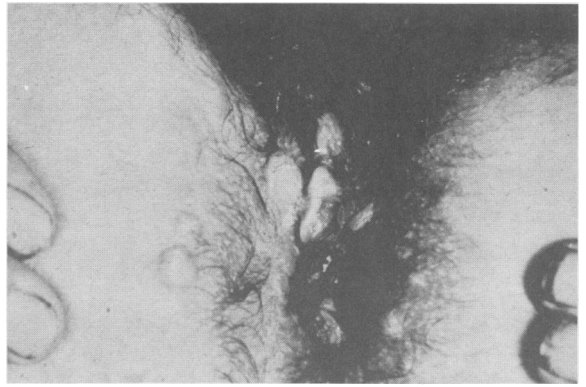

Figure 9.—Condylomata lata

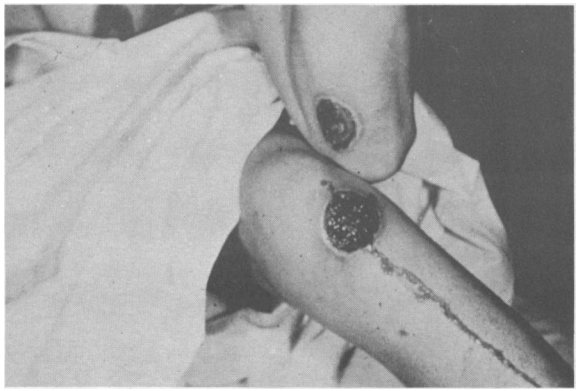

Figure 10.—Lues maligna

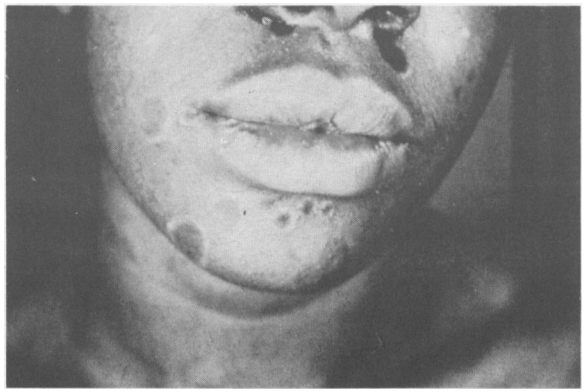

Figure 11.—Annular syphilid

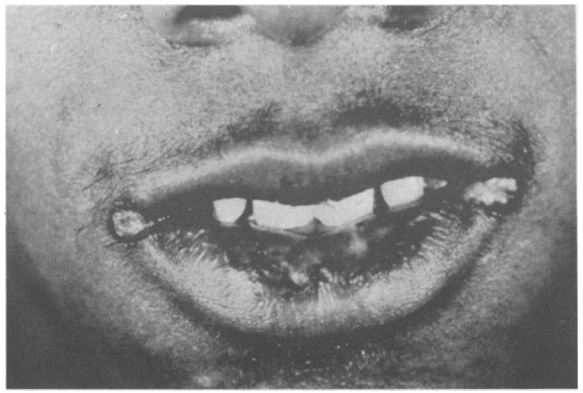

Figure 12.—Split papules

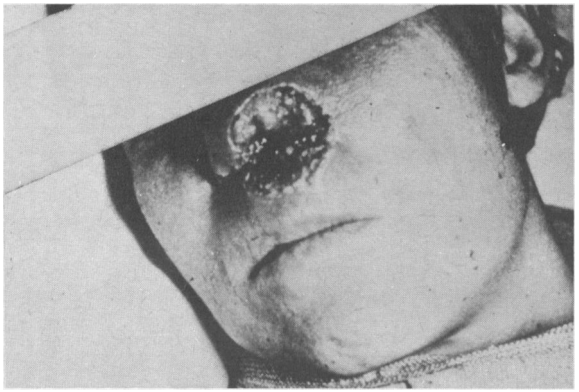

Figure 13.—Gumma, nose

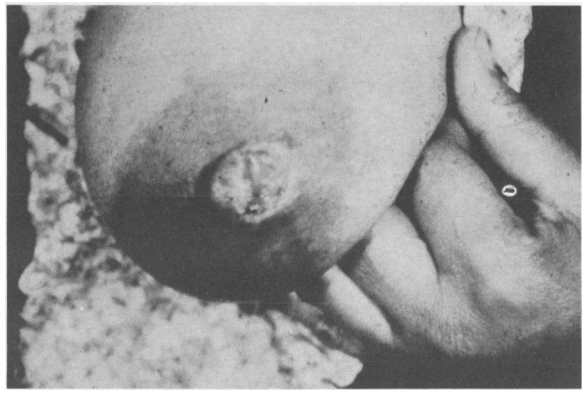

Figure 14.—Gumma, breast

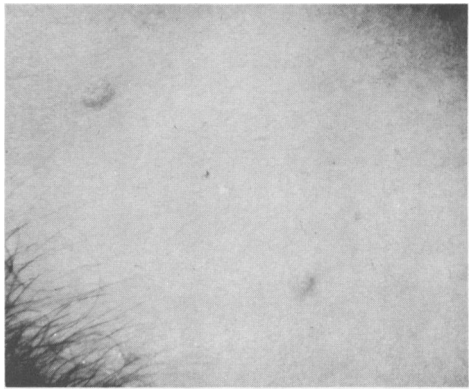

Figure 15.—Papulonodular syphilid

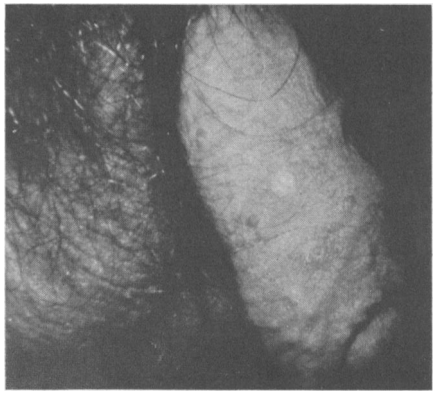

Figure 16.—Papulonodular syphilid

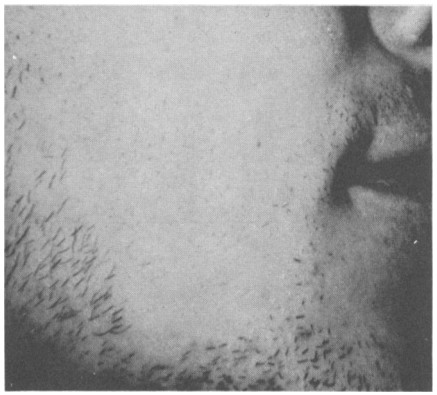

Figure 17.—Alopecia of beard

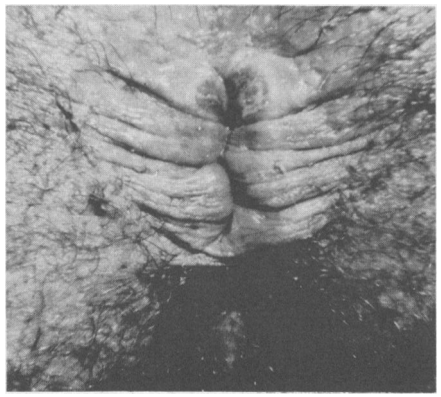

Figure 18.—Secondary syphilis, anal lesion

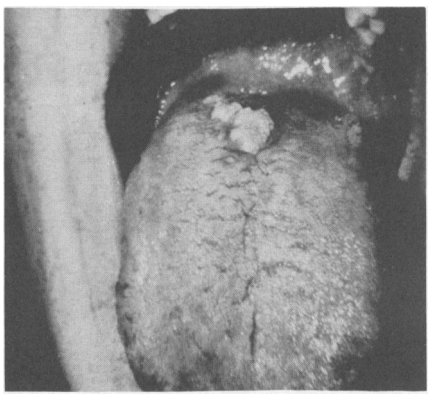

Figure 19.—Keratotic syphilid of tongue

first complement fixation test. Paul Ehrlich in 1910 introduced arsphenamine, also called Salvarsan® or 606, which of course was the main form of syphilitic therapy until penicillin was discovered by Fleming in 1943. Julius von Wagner-Jauregg, about 1917, introduced the concept of fever therapy.

A Key to Fever Therapy in an Old Report

It is of interest that when de Isla wrote his treatise on the serpentine disease in 1539, he observed that patients who had malaria, and high fevers from that disease, appeared not to get syphilis. Unfortunately, this observation was totally lost for the next 400 years. Von Wagner-Jauregg again discovered that if one elevates a patient's temperature to about 104°F for a time, Treponema pallidum are killed. Following von Wagner-Jauregg's work, treatment of patients in an electronic cabinet or by wrapping them in hot blankets was widely used in the treatment of syphilis from about 1920 until the mid 40's. One reason this classical treatment should be of interest to those in this audience is that Doctors Edward Levine, Norman Epstein and Francis Torrey of our Department of Dermatology used the treatment successfully in a number of patients, and the University of California, San Francisco, was one of the major institutions on the West Coast where fever therapy was used in the treatment of syphilis. All of this changed in 1943 with the introduction of penicillin, and many authorities felt that the young shepherd, Syphilus, had finally escaped further punishment from vengeful gods; but, unhappily, as I have mentioned, this has not been the case.

Let us look at some of the classical lesions of the disease. Figure 1 shows a classical indurated, ulcerated, nonpainful chancre such as is usually associated with lymphadenopathy. This lesion can be anywhere. It can present on the shaft or glans of the penis, on the mons pubis, labia, or cervix. More rarely, the lesion is discovered on the tongue or a tonsillar pillar, or a finger. Patients will frequently not seek medical attention because the ulcer is not painful and they cannot imagine that a nonpainful lesion can be harmful. The lesion seen in Figure 2 may cause some difficulty with urination. You may not find a large node with such lesions because lymph drainage is back to the periaortic nodes. However, on darkfield examination, as with all primary and secondary lesions of syphilis, the lesion will be teeming with treponemes. Figure 3 is a picture of a chancre on a finger, but I think that few of us in this room would think of syphilis as the first diagnosis. A giveaway may be the fact that the lesion is not painful and, of course, the patient will have a large axillary or an epitrochlear node. Perianal and anal lesions occur commonly in both males and females and even, (Figure 4) unfortunately, in children. The lesion shown in Figure 5 could be easily confused with herpes simplex. Even the large submandibular node seen here, which in this case is a syphilitic bubo, can also be seen with herpes. If the patient does not give a classical history of recurrent herpes simplex, a darkfield examination on this lesion is mandatory.

The Mimicry of Secondary Syphilis

The lesions of secondary syphilis provide a wide clinical spectrum and are a paradise for the morphological dermatologist. The disease in this stage can take on almost any appearance (Figure 6). In Figure 7 you see the rather classical papulosquamous lesions of secondary syphilis. Patients with this condition will usually tell you that they have mild sore throat and a little hoarseness. Frequently, they will have noted a mild upper respiratory tract infection. Many internists on seeing a patient with a mild evanescent rash, a little upper respiratory tract infection, malaise, and lymphadenopathy might well consider mononucleosis as their first diagnosis. Secondary syphilis must be considered in your differentiations, and a specimen for a VDRL test must be drawn. Papulosquamous lesions may be confused with pityriasis rosea or even psoriasis. The papulopustular form of syphilis is not seen very frequently today but is apparently the type of pox lesion that was described by de Villalobos and others in the early 1500's. Palmar and plantar lesions with hyperkeratotic papules are very common in secondary syphilis. One point that helps in clinical differentiation of pityriasis rosea from secondary syphilis is the fact that plantar and palmar lesions are rare in pityriasis rosea and common in syphilis. However, you should always get a VDRL test to confirm your clinical impression.

The papular nodular lesions in syphilis (Figure 8) may be slightly tender with pressure and usually the eruption does not itch. Condylomata lata (Figure 9) are easily confused with condylomata acuminata. Again, this is a lesion of sec-

ondary syphilis, and the patient will have a high VDRL titer. These lesions are teeming with treponemes, which can be easily recovered for darkfield examination.

The drier lesions on the body produce a greater problem, but frequently organisms can be demonstrated in these lesions if a saline sponge is used to prehydrate the lesion for ten to fifteen minutes. The top of the lesion is then lightly debrided with a dry swab, pressure is applied on the sides of the lesion, and sera is collected for examination.

We saw a young patient here some years ago who was suffering from a rare but well described variant of secondary syphilis which has been called lues maligna (Figure 10). In this condition, large ulcerative lesions are usually seen. The disease is one of late secondary lues and commonly lasts for weeks to months. The patient had consulted two or three physicians, who had not come to a correct diagnosis. He then became a recluse and turned to Christian Science. When the disease continued to spread, he came to this hospital where the proper diagnosis was made by Dr. D. A. Fisher, and the patient was treated with penicillin.

The annular lesions of secondary syphilis (Figure 11) are seen almost exclusively in black patients. If these lesions occur at the corner of the nose or the edge of the mouth, they are referred to as split papules (Figure 12). If you see such a lesion, syphilis is probably a much more likely diagnosis than cheilitis due to a vitamin deficiency. Figure 13 shows a tertiary lesion of syphilis destroying the nasal septum and Figure 14 shows a similar lesion on a breast. I think most clinicians would be far more concerned about carcinoma, but do not let your concern for that disease dull your diagnostic acumen to the point that you neglect to order a VDRL and FTA-ABS to rule out tertiary syphilis.

The pictures that you have just seen are some classical photographs of primary and secondary syphilis distributed by the Public Health Service. I would like to end this dissertation by showing you pictures from four or five cases that I have seen over the last year or two to indicate how we are seeing syphilis in this community today. The lesions seen in Figures 15 and 16 had been present for a few days and they itched slightly. The patient was concerned about scabies. As you see, the lesions have no scales such as appear in classical secondary syphilis. These small papulonodules were present over most of the body but particularly in the inguinal area and on the penis. The VDRL reaction was 1:64 and these are lesions of secondary syphilis.

Figure 17 shows the face of a young man who sought medical advice because of alopecia of the beard area. Most of us would immediately make a diagnosis of alopecia areata and advise the patient that little or nothing can be done to treat the condition. But the patient said in his history that at the time this loss of hair in the beard area had developed, he had begun to have an unusual, nonpruritic eruption on the chest, which he had attributed to a fungus infection. Examination revealed an erythematous macular eruption which could in fact have been confused with tinea versicolor. On the patient's penis, there was a small papulonodular lesion and the patient's VDRL reaction was 1:64.

Another patient had seen his family doctor for a lesion in the anal area which had been present for about two weeks. A diagnosis of hemorrhoids had been made and the patient had been advised to use phenylmercuric nitrate (Preparation H®). On examination a tender, ulcerative lesion with a large fissure running diagonally through the center was seen (Figure 18). Darkfield examination of the lesion was positive for syphilis, and one would certainly consider the diagnosis of primary syphilis, but on further examination a 3 to 4 mm papulonodular lesion was seen on the glans penis. The patient's VDRL was 1:64, and he too had secondary syphilis.

The next case presented a fascinating problem a few years back. The patient, a young man, consulted a physician because of lymphadenopathy. He was told that he had mononucleosis. A heterophile test was done, and the physician later reported to the patient that his case was most unusual because the result of the test was negative. The boy was treated conservatively, and when he did not improve he consulted another physician who also told him that he had mononucleosis. The second physician also ordered a heterophile test and he, too, reported to the patient that the test was negative. Finally, a keratotic lesion (Figure 19) developed on the patient's tongue. The lesion was quite firm and did not scrape off easily. The patient presented himself to the University of California emergency room, where a diagnosis of mononucleosis with thrush was made. For treatment of thrush he was referred to the dermatology clinic, and there a darkfield examination was done. This oral lesion was teeming with treponemes. The VDRL titer was high. The patient was

treated with penicillin. Here was a case of secondary syphilis misdiagnosed by three physicians as mononucleosis.

Two points I would have you keep in mind. The first is that syphilis is occurring in this community and that it is occurring with great frequency. The second is that the disease has slowly changed through the centuries and apparently is becoming more insidious and frequently obtuse in its initial presentation. Certainly, if any physicians in America have an opportunity to diagnose and treat syphilis, it is those of you seated in this audience this morning, and I hope for your sake and the sake of your patients that you do not miss this opportunity.

Trade and Generic Names of Drugs

Salvarsan® ..... Arsphenamine
Preparation H® ..... Phenylmercuric nitrate

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Selection of Shoes for Growing Feet

With regard to shoes and footgear in the growing foot, the critical consideration is that the shoe fit properly. The shoe should be long enough to allow an adult thumb breadth between the end of the great toe and the end of the toe box of the shoe when the child is standing. It should also be wide enough so that, when the fifth metatarsal is pushed against the lateral edge of the upper part of the shoe, approximately 1/8th to 3/16th inch of slack can be felt to overlie the first metatarsal head on the medial side of the shoe. The heel should not ride up and down excessively as the child walks; and with regard to material, I think that soft leather or canvas shoes (such things as sneakers) are probably preferable to rigid counters and hard leathers. The box-toe style of shoe—that is to say, an *un*pointed shoe—is the only proper footgear at any age. More problems have been caused by high-heeled pumps with the pointed shoe, which was formerly popular among young women, than by any shoes made for children.

A question frequently asked of pediatricians or family practitioners is about the time when the baby should be given his first pair of shoes. There is no good evidence that shoes do anything for the feet other than protect them, and hence it probably makes no sense to buy shoes until protection from the cold ground or glass or rocks underfoot becomes essential. Walking barefoot in the house, or with a soft moccasin type of shoe, is probably best when the infant begins to walk. Elevated heels, that are currently in style, are not a good idea; but, by and large, American childrens' shoe styles are good. Another frequently asked question is whether high-top or low-top shoes are preferable. I think it really makes no difference from the standpoint of the foot. High lace-up shoes are harder for the baby to take off and hence probably are better for mama's back, but make very little difference to the development of the baby's foot. Normal toddlers do not have significantly weak ankles and do not need the support of a high-top shoe about the ankle.

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